Published by Elsevier Inc.

ISSN 0735-1097/04/$30.00
doi:10.1016/j.jacc.2003.08.031

Myocardial Infarction After Percutaneous Transluminal Septal Myocardial Ablation in Hypertrophic Obstructive Cardiomyopathy: Evaluation by Contrast-Enhanced Magnetic Resonance Imaging

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OBJECTIVES The aim of this study was to evaluate myocardial infarction induced by percutaneous transluminal septal myocardial ablation (PTSMA) in symptomatic patients with hypertrophic obstructive cardiomyopathy using contrast-enhanced (CE) magnetic resonance imaging (MRI).

BACKGROUND Contrast-enhanced MRI delineates the extent of myocardial infarction in coronary artery disease, but its role in ethanol-induced infarction has not been established.

METHODS Cine and CE MRI were performed before and one month after PTSMA in 24 patients. Size and location of the induced infarction were related to left ventricular (LV) mass reduction, enzyme release, volume of ethanol administered, LV outflow tract gradient reduction, and coronary ablation site.

RESULTS One month after PTSMA, regional hyperenhancement was visualized in the basal interventricular septum in all patients. Mean infarction size was 20 ± 9 g, corresponding to 10 ± 5% and 31 ± 16% of total LV and septal mass, respectively. Total LV mass decreased from 219 ± 64 to 205 ± 64 g ($p < 0.01$), and septal mass from 76 ± 25 to 68 ± 22 g ($p < 0.01$). Total LV mass reduction exceeded septal mass reduction ($p < 0.01$). Infarction size correlated with peak creatine phosphokinase-MB ($\beta = 0.67$, $p < 0.01$), volume of ethanol administered ($\beta = 0.47$, $p = 0.02$), total LV and septal mass reduction ($\beta = 0.50$, $p = 0.02$; $\beta = 0.73$, $p < 0.01$), and gradient reduction ($\beta = 0.63$, $p < 0.01$). Seven patients with exclusively right-sided septal infarction had smaller infarction size and less gradient reduction than remaining patients with left-sided or transmural infarction ($p < 0.01$). In five of these, PTSMA was performed distal in the target artery.

CONCLUSIONS Contrast-enhanced MRI allowed detailed evaluation of size and location of septal myocardial infarction induced by PTSMA. Infarction size correlated well with clinical indexes of infarct size. (J Am Coll Cardiol 2004;43:27–34) © 2004 by the American College of Cardiology Foundation

Hypertrophic cardiomyopathy is a heterogeneous disease characterized by myocardial hypertrophy in the absence of any other systemic or cardiac disease, with predominant involvement of the interventricular septum (IVS) (1,2). Approximately 25% of the patients have a dynamic left ventricular outflow tract (LVOT) obstruction caused by a narrowed LVOT and abnormal systolic anterior motion of the mitral valve.

Treatment strategies in patients with hypertrophic obstructive cardiomyopathy (HOCM) who remain symptomatic despite optimal medication (beta-adrenergic blocking agents, verapamil, and disopyramide) include surgical (septal myotomy-myectomy) and nonsurgical procedures such as dual-chamber pacing and percutaneous transluminal septal myocardial ablation (PTSMA) (3–9); PTSMA is a recently developed procedure, which consists of artificially inducing a localized myocardial infarct by ethanol infusion into septal branches of the left anterior descending coronary artery (7–10). Scarring and thinning of the IVS results in widening of the LVOT, a decrease of the pressure gradient, and symptomatic improvement (11–13).

The final outcome after PTSMA is thought to depend on size and location of the inflicted infarction. Infarcts that are too small or are located outside the target area may not achieve the necessary reduction in LVOT gradient. Large infarcts may cause potentially hazardous conduction abnormalities or ventricular arrhythmias. Echocardiography with intracoronary contrast injection is commonly used during the procedure to guide the selection of the appropriate septal branch, but this technique does not allow visualization of the infarction site at follow-up. Myocardial perfusion defects by single-photon emission computed tomographic myocardial scintigraphy at six weeks after PTSMA correlated with the target area for ablation defined by contrast echocardiography (14,15). However, the spatial resolution

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Manuscript received February 27, 2003; revised manuscript received August 1, 2003, accepted August 5, 2003.

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Abbreviations and Acronyms

CE = contrast-enhanced
CK = creatine phosphokinase
HOCM = hypertrophic obstructive cardiomyopathy
IVS = interventricular septum
LV = left ventricle/ventricular
LVOT = left ventricular outflow tract
MRI = magnetic resonance imaging
NYHA = New York Heart Association
PTSMA = percutaneous transluminal septal myocardial ablation

of this technique is not sufficient to allow transmural evaluation.

In patients with ischemic heart disease, contrast-enhanced (CE) magnetic resonance imaging (MRI) accurately delineates infarcted, irreversibly damaged myocardium, both in the (sub-)acute and in the chronic phases (16,17). The high spatial resolution of CE MRI would allow detailed evaluation of the ethanol-induced infarction. Contrary to contrast echocardiography, CE MRI cannot be used during the ablation procedure, but it may provide important feedback by accurately delineating the ultimate size and location of the infarction. Although CE MRI is capable of visualizing PTSMA-related infarction, its exact significance still needs to be established (18).

The purpose of the study was to evaluate myocardial infarction size and location induced by PTSMA in patients with HOCM using CE MRI, and correlate the findings to procedural and infarct-related parameters and early clinical outcome.

METHODS

Patients. The study protocol was approved by the Committee on Research Involving Human Subjects and the Medical Ethics Committee of the VU University Medical Center Amsterdam. All patients with HOCM who were scheduled for PTSMA were eligible for MRI. The indication for PTSMA was based on a significant LVOT gradient as documented by echocardiography and New York Heart Association (NYHA) functional class II to IV despite medical treatment. Exclusion criteria were any absolute or relative contraindication to MRI (e.g., pacemaker, claustrophobia), atrial fibrillation, or failure to give informed consent. Twenty-eight patients were initially enrolled. Four patients were excluded from the final analysis: three required pacemaker-implantation during or after PTSMA, and one declined to return for the follow-up examination. The baseline characteristics of the remaining 24 patients are listed in Table 1.

Echocardiography. Baseline echocardiographic measurements of IVS and posterior wall thickness, left ventricular (LV) end-diastolic, and left atrial end-systolic dimensions are listed in Table 1. The LVOT pressure gradient was documented by Doppler echocardiography at baseline and one month after PTSMA. A pressure gradient ≥50 mm Hg at rest was considered to be significant. Three patients had a resting gradient <50 mm Hg, and provocation was applied both at baseline and follow-up using dobutamine echocardiography and the Valsalva maneuver in two and one patients, respectively. An increase of the pressure gradient during provocation to ≥50 mm Hg was considered significant.

Table 1. Patient Characteristics

Characteristics	Patients (n = 24)
Age (yrs)	52 ± 15
Men/women	11/13
NYHA functional class	2.9 ± 0.4
II/III/IV	3/20/1
Symptoms	
Dyspnea	22
Angina	9
Syncope	6
Family history	
HCM/sudden death	14/7
Medication	
Beta-blockers	16
Calcium-antagonists	12
Diuretics	2
Echocardiographic parameters at baseline	
LVOT gradient, mm Hg	87 ± 22
IVS thickness, cm	2.1 ± 0.4
Posterior wall thickness, cm	1.3 ± 0.2
LVEDD, cm	4.5 ± 0.5
LAESD, cm	4.9 ± 0.4

Values expressed as mean ± SD.
HCM = hypertrophic cardiomyopathy; IVS = interventricular septum; LV = left ventricular; LAESD = left atrial end-systolic diameter; LVEDD = left ventricular end-diastolic diameter; LVOT = left ventricular outflow tract; NYHA = New York Heart Association.

PTSMA procedure. All patients underwent PTSMA in one of two major referral centers in the Netherlands (Thoraxcenter Erasmus Medical Center, Rotterdam, 16 patients; St. Antonius Hospital, Nieuwegein, 8 patients).

Using a standard Judkins technique, an A6F pacemaker lead was placed in the right ventricle, an A6F pigtail catheter was positioned into the LV, and an A7F Judkins guiding catheter in the ascending aorta. The LVOT pressure gradient was continuously monitored throughout the whole procedure. After initial angiography for localizing the origin of the septal perforating arteries, a 1.5-2.5 × 10-mm balloon catheter was introduced over a 0.014-inch guidewire into the target perforator artery and inflated. Contrast (Levovist, Schering AG, Berlin, Germany) was then injected through the balloon catheter shaft during simultaneous registration of transthoracic two-dimensional echocardiography to determine the part of the myocardium supplied by the targeted septal artery. If no leakage of contrast occurred into the LV cavity, ethanol was slowly (1 ml/min) injected up to a maximum of 5 ml. Five minutes after ethanol injection, the balloon was deflated and coronary arteriography repeated. A successful procedure was

defined as the reduction in LVOT pressure gradient of ≥50% of baseline. If the results were not satisfactory, the whole procedure, including echocardiography-contrast injection, was repeated in another septal branch.

The total volume of ethanol injected into perforating arteries of the left anterior descending coronary artery during the procedure was documented. Plasma creatine phosphokinase (CK) and CK-MB fraction levels were determined before and every 6 h after the procedure during a 24-h period.

MRI. Magnetic resonance imaging was performed 11 ± 11 days before and 32 ± 9 days after PTSMA on a 1.5 Tesla clinical scanner (Sonata, Siemens, Erlangen, Germany), using a four-element phased-array body radiofrequency receiver coil. All images were acquired with electrocardiogram gating and during repeated breath-holds of 10 to 15 s, depending on heart rate. After localizing scouts, cine images were acquired using a segmented steady-state-free precession sequence in three long-axis views (two-, three-, and four-chamber view) and in multiple short-axis views every 10 mm, covering the whole LV from base to apex. Scan parameters were: temporal resolution 34 ms, TR 3.0 ms, TE 1.5 ms, typical voxel size $1.4 \times 1.8 \times 5$ mm^3.

Contrast-enhanced images were acquired 15 to 20 min after intravenous administration of 0.2 mmol/kg gadolinium-DTPA in the same views used in cine MRI, using a two-dimensional segmented inversion-recovery prepared gradient-echocardiography sequence (TE 4.4 ms, TR 9.8 ms, inversion time 250 to 300 ms, typical voxel size $1.3 \times 1.6 \times 5$ mm^3) (19,20). Contrast-enhanced images were acquired in all patients at follow-up MRI. As small patchy areas of hyperenhancement were noted in the IVS outside the infarcted region, CE imaging was added to the baseline protocol in patients 11 to 24.

Data analysis. Contrast-enhanced images, cine images, and catheterization data were analyzed separately, and all observers were blinded to the results of the other investigations.

ANALYSIS OF CE IMAGES. Contrast-to-noise ratio of the hyperenhanced area versus a remote nonenhanced myocardial area was measured on the short-axis slice demonstrating the largest area of hyperenhancement. Contrast-to-noise ratio was calculated using regions of interest and defined as: $(SI_{hyperenhanced} - SI_{remote})$/noise, where SI is signal intensity, and noise is expressed as the SI SD in a background region of interest. Myocardial infarction size after PTSMA was measured by manual tracing of the hyperenhanced areas. The hyperenhanced area was defined as the area within the septal myocardium with pixel SI values >4 SD of remote, nonenhanced myocardium. Central dark zones within the area of hyperenhancement were included.

ANALYSIS OF CINE IMAGES. Left ventricular parameters, including end-diastolic volume, end-systolic volume, ejection fraction, total and septal myocardial mass, and maximum end-diastolic IVS thickness at the infarct site and posterior wall thickness were quantified using the MASS software package (MEDIS, Leiden, the Netherlands). Endocardial and epicardial borders were outlined manually in end-diastolic and end-systolic frames of all short-axis slices. Papillary muscles were included in the assessment of LV mass. The IVS was defined as the myocardium between the anterior and posterior junctions of the right ventricle to the LV.

ANALYSIS OF CORONARY ARTERIOGRAMS. All coronary arteriograms were analyzed and scored in consensus by two experienced interventional cardiologists (J.M.t.B., J.V.), who were blinded to the magnetic resonance results. The number of septal perforating arteries, the target artery, and the ablation site within the target artery were registered.

Statistical analysis. Results are expressed as mean ± SD. Paired *t* tests were used to evaluate the changes of LV mass and volumes after PTSMA. Linear regression analysis was used to analyze the relationship between myocardial infarction size (outcome variable) and cardiac enzymes, the volume of ethanol administered during the ablation procedure, total LV and septal mass reduction, and LVOT gradient reduction. The analyses were adjusted for age, and the results are presented as age-adjusted standardized regression coefficients (β), which can be interpreted as partial correlation coefficients. The Mann-Whitney *U* test was used to evaluate the correlation between different infarction locations and myocardial infarction size, the volume of ethanol administered, and LVOT gradient reduction.

All statistical analyses were performed with SPSS (version 11.0), and significance was set at a p value ≤0.05.

RESULTS

Mean volume of ethanol injected was 3.3 ± 1.7 ml, and mean peak CK and CK-MB release was 1,592 ± 775 U/l and 198 ± 84 U/l, respectively. One month after PTSMA, mean LVOT pressure gradient decreased from 87 ± 22 mm Hg to 23 ± 29 mm Hg ($p < 0.01$), and mean NYHA class improved significantly from 2.9 ± 0.3 to 1.7 ± 0.6 ($p < 0.01$). During the follow-up period, none of the patients experienced syncope, and there were no documented ventricular arrhythmias.

CE MRI at baseline. In 14 patients, CE imaging was performed before PTSMA. In 10 patients, small patchy areas of hyperenhanced myocardium were observed in the IVS, located centrally in the ventricular wall and predominantly at the junctions of the right and LV free walls. The number of focal areas of hyperenhancement per patient was 4 ± 1.5, representing an average mass of 0.5 ± 0.4 g per area. The contrast-to-noise ratio of these areas was 7 ± 4. Figure 1A shows an example of focal contrast-enhancement in a patient before PTSMA.

CE MRI after PTSMA—infarct size. At follow-up, a clearly demarcated area of hyperenhancement was visualized in the basal part of the IVS in all patients. No patient had evidence of infarction-related hyperenhancement outside

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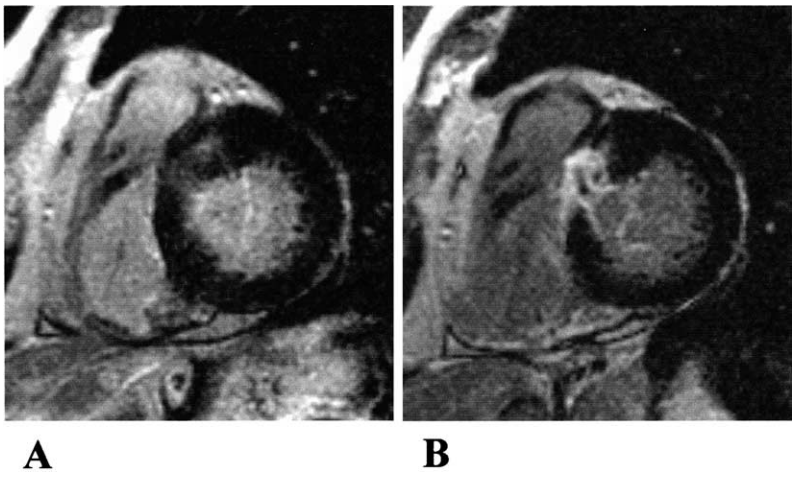

Figure 1. Contrast-enhanced short-axis images before **(A)** and after percutaneous transluminal septal myocardial ablation (PTSMA) **(B)** in a patient with hypertrophic obstructive cardiomyopathy. Before PTSMA, a slightly enhanced myocardial region is apparent in the anterobasal wall of the interventricular septum. After PTSMA, the ethanol-induced infarction can be seen as a clearly demarcated area of hyperenhancement.

the target area. The contrast-to-noise ratio of hyperenhanced areas was 26 ± 7. Mean myocardial infarction size was 20 ± 9 g (range, 5 to 41 g), involving 10 ± 5% of the post-ablation total LV mass and 31 ± 16% of the septal myocardial mass. Figures 1B and 2 show examples of ethanol-induced infarctions.

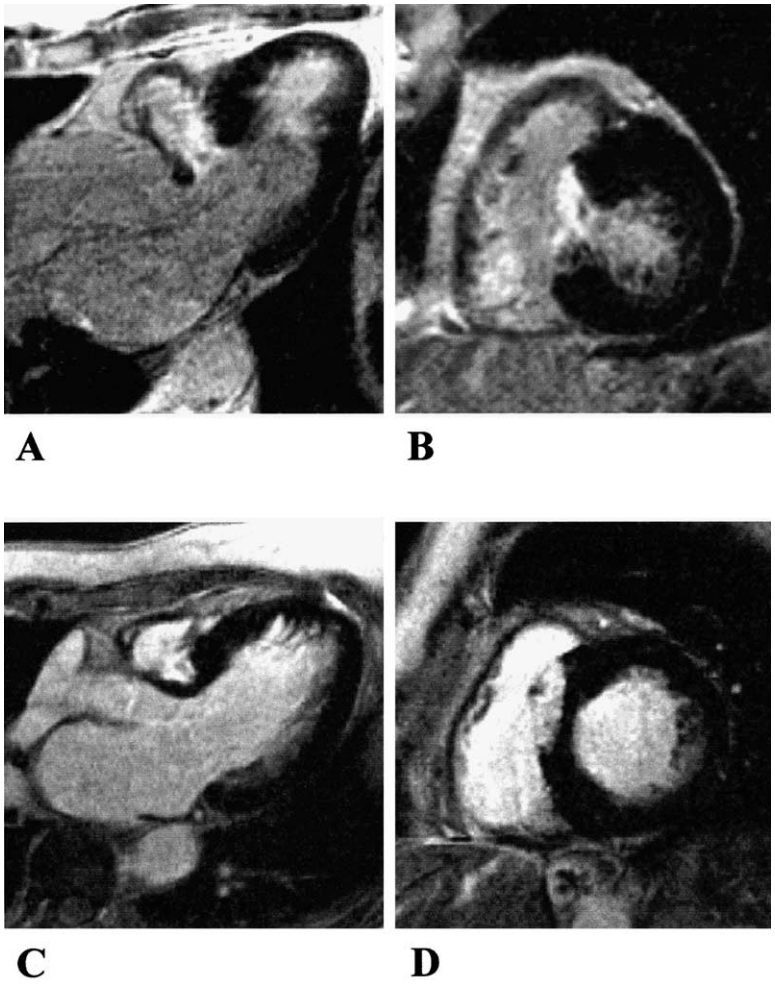

Figure 2. Contrast-enhanced images 20 min after intravascular administration of gadolinium-DTPA in two patients with hypertrophic obstructive cardiomyopathy one month after percutaneous transluminal septal myocardial ablation. **(A, B)** Three-chamber view and short-axis view in a patient with transmural septal infarction. **(C, D)** Comparable views in a patient with myocardial infarction located exclusively on the right ventricular side of the interventricular septum.

Table 2. Cine MRI and Clinical Parameters Before and 1 Month After PTSMA

	Baseline	Follow-Up	p Value
Cine MRI parameters (cm)			
IVS thickness at infarct site (cm)	2.1 ± 0.4	1.6 ± 0.5	$p < 0.01$
LV PW thickness (cm)	0.8 ± 0.2	0.8 ± 0.1	NS
End-diastolic volume (ml)	153 ± 41	154 ± 38	NS
End-systolic volume (ml)	47 ± 14	53 ± 17	$p = 0.03$
LV ejection fraction (%)	69 ± 5	67 ± 5	$p = 0.01$
Total LV mass (g)	219 ± 64	205 ± 64	$p < 0.01$
Septal mass (g)	76 ± 25	68 ± 22	$p < 0.01$
Clinical parameters			
NYHA functional class	2.9 ± 0.4	1.7 ± 0.6	$p < 0.01$
LVOT gradient, mm Hg	87 ± 22	23 ± 29	$p < 0.01$

Values expressed as mean ± SD.

IVS = interventricular septum; LV = left ventricular; LVOT = left ventricular outflow tract; MRI = magnetic resonance imaging; NS = statistically not significant; NYHA = New York Heart Association; PTSMA = percutaneous transluminal septal myocardial ablation; PW = posterior wall.

Changes in LV mass and volumes. End-diastolic IVS thickness measured at the site of infarction decreased from 2.1 ± 0.4 cm at baseline to 1.6 ± 0.5 cm at follow-up ($p < 0.01$). Total LV myocardial mass decreased significantly from 219 ± 64 g at baseline to 205 ± 64 g after PTSMA ($p < 0.001$). Septal myocardial mass decreased from 76 ± 25 g pre-PTSMA to 68 ± 22 g post-PTSMA ($p < 0.01$). The reduction in total LV mass was larger than that in septal mass only ($p < 0.01$). The reduction in remote myocardial mass (i.e., excluding the septal mass) was statistically significant ($p < 0.01$). At follow-up, LV end-diastolic volumes were unchanged (153 ± 41 ml vs. 154 ± 38 ml). A significant increase of LV end-systolic volumes was observed (47 ± 14 ml vs. 53 ± 17 ml; $p = 0.03$), with a concurrent decrease in LV ejection fraction (69 ± 5% vs. 67 ± 5%; $p = 0.01$). Changes of LV parameters are summarized in Table 2.

Correlation between infarct size and other parameters. Linear regression analysis showed significant associations between myocardial infarction size and both peak CK and CK-MB after PTSMA. The age-adjusted standardized regression coefficients (β) were 0.53 ($p = 0.01$) and 0.67 ($p < 0.01$), respectively (Fig. 3). Myocardial infarction size also correlated with the volume of ethanol administered ($\beta = 0.47$, $p = 0.02$; Fig. 4), total and septal mass reduction ($\beta = 0.50$, $p = 0.02$; $\beta = 0.73$, $p < 0.01$, respectively), and the reduction in the LVOT pressure gradient measured by Doppler echocardiography ($\beta = 0.63$, $p < 0.01$; Fig. 3).

CE MRI after PTSMA—infarct location. The area of hyperenhancement was located exclusively on the right ventricular side of the IVS in seven patients, exclusively on the LV side of the IVS in two patients, and extended transmurally throughout the IVS in 15 patients. Examples of a patient with transmural extent and a patient with exclusively right-sided location of septal infarction are shown in Figure 2. In the seven patients with exclusively right-sided hyperenhancement infarct size, septal mass reduction and reduction in LVOT gradient were smaller (10 ± 4 g vs. 23 ± 8 g; 2.5 ± 1.3 g vs. 9.4 ± 4.0; and 30 ± 28 mm Hg vs. 78 ± 22 mm Hg; all $p < 0.01$). The volume of ethanol infused during the ablation procedure tended to be lower (2.6 ± 1.2 ml vs. 3.6 ± 1.8 ml, $p = 0.19$). In these patients, mean NYHA functional class improved from 3.1 ± 0.4 to 2.3 ± 0.5 and, in the transmurally infarcted patients, from 2.8 ± 0.4 to 1.4 ± 0.5. The two patients without symptomatic improvement had a right-sided location of the septal infarction without reduc-

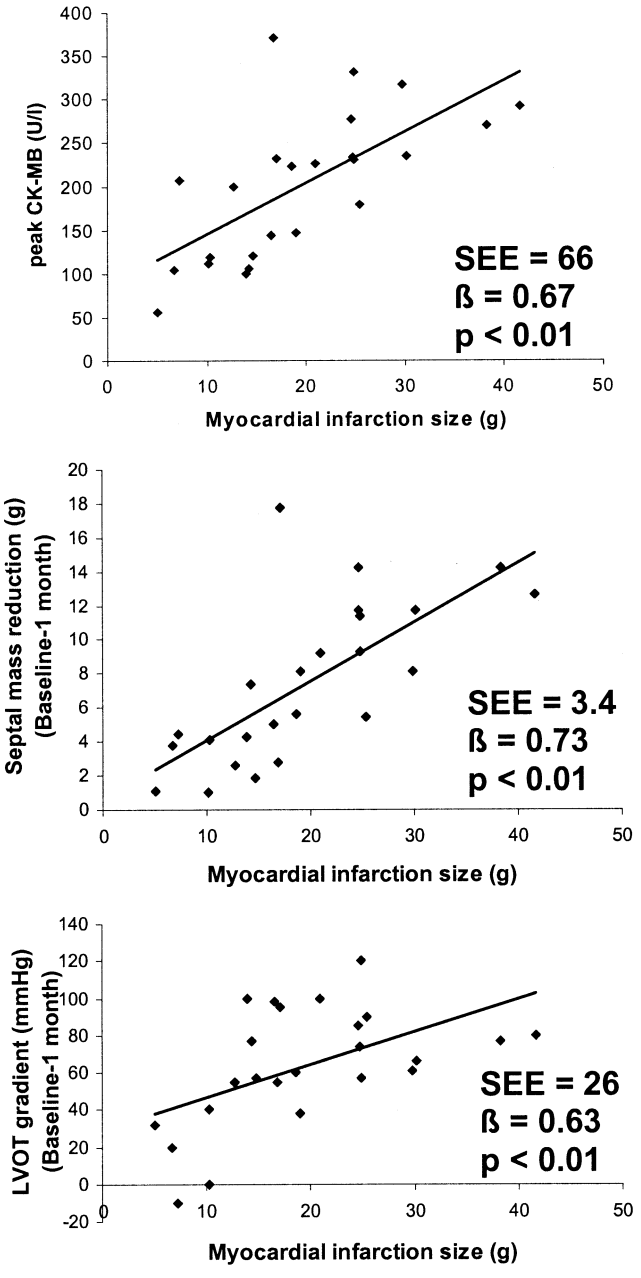

Figure 3. Correlation of myocardial infarction size and peak creatine phosphokinase (CK)-MB, reduction in septal mass, and the reduction in the left ventricular outflow tract (LVOT) pressure gradient after percutaneous transluminal septal myocardial ablation. The age-adjusted standardized regression coefficient (β) and the significance (p value) are given. SEE = standard error of the estimate.

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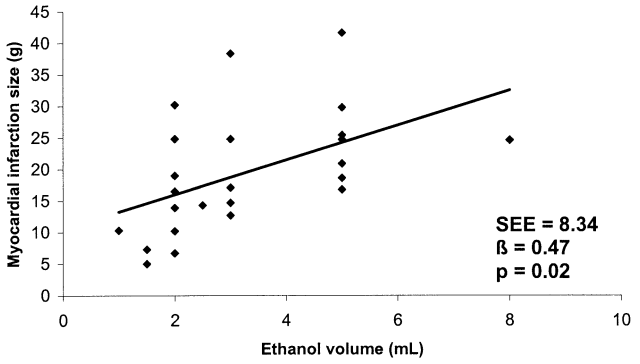

Figure 4. Correlation of the volume of ethanol (ml) injected and myocardial infarction size (g) after percutaneous transluminal septal myocardial ablation. The age-adjusted standardized regression coefficient (β) and the significance (p value) are given.

tion in the LVOT gradient. The infarction size of these two patients was 7.3 g and 10.2 g, respectively.

Correlation between infarct location and coronary ablation site. The average number of septal perforating arteries per patient was 3.6 ± 0.8 (range, 2 to 6). The target artery was the first, second, and third perforator in 13, 11, and 2 patients, respectively. In two patients, ethanol infusion was repeated after 20 min in a second artery, because of insufficient gradient reduction after the first ethanol infusion.

In the 15 patients with transmural infarction and the two patients with left-sided infarction of the IVS, PTSMA was performed proximal to the first bifurcation of the target artery. In the seven patients with exclusively right-sided hyperenhancement of the IVS, PTSMA was performed distal to the first bifurcation of the target artery in four and more distal than usual in a target artery without side branches in one.

DISCUSSION

Our findings demonstrate that CE MRI allows the detailed evaluation of the PTSMA-induced myocardial infarction in symptomatic patients with HOCM. The quantified infarction size was significantly correlated to cardiac enzyme release, volume of ethanol administered, total LV and septal mass reduction, and LVOT pressure gradient reduction. Also, patients with an exclusively right-sided location of the infarction within the IVS had significantly smaller infarction size and less gradient reduction.

Mechanism of hyperenhancement. After coronary artery occlusion, the extracellular contrast agent gadolinium-DTPA accumulates in infarcted regions of the myocardium that are necrotic and irreversibly damaged (21). In (sub)-acute infarction, this is the result of altered wash-in and wash-out characteristics and an increased volume of distribution caused by myocyte membrane disrupture (20,22). The mechanism of contrast agent accumulation in PTSMA-induced infarctions may be similar, due to the direct toxic, osmotic, and thrombotic effects of ethanol spreading throughout the myocardial tissue (23). Because our data were acquired one month after PTSMA, at a time when the infarcted myocardium is likely to have entered the chronic phase, other mechanisms like passive diffusion of the contrast agent within enlarged interstitial compartments of the collagen matrix in fibrous scar may have played a role (21). This mechanism may also explain the presence of patchy focal areas of hyperenhancement that we found in the majority of patients that underwent CE MRI before PTSMA. Choudhury et al. (24) recently reported the presence of multiple foci of patchy hyperenhancement in the majority of a group of asymptomatic patients with HCM. They found that these areas were predominantly located in the middle third of the ventricular wall, at the junction of the septum and right ventricular free wall, which is similar to our findings. The clinical significance of these areas is unknown, but they may act as a substrate for the development of ventricular arrhythmias (25).

Size and location of myocardial infarction. The range of myocardial infarction size was large (5 to 41 g). Factors that may influence infarction size include differences in septal coronary anatomy, position of the inflated balloon within the target artery during ethanol infusion, and volume of ethanol administered.

In seven patients, infarction was located exclusively on the right ventricular side of the septum. These patients had smaller infarction size and less reduction in LVOT gradient, and two patients reported no symptomatic improvement. In contrast with patients with transmural or exclusively left-sided septal infarction, balloon position during ethanol infusion was frequently distal to a bifurcation. Currently available autopsy data of septal coronary anatomy do not provide a conclusive explanation for the differences in location of the ablation injury (26). Our findings suggest that an exclusively right-sided septal infarction may be related to the ablation site.

Effect of PTSMA on LV mass and volumes. Previous echocardiographic studies have demonstrated a significant reduction in LV mass one year after PTSMA. This was not only due to thinning of septal myocardium, but also to a decrease of wall thickness throughout the LV circumference (27,28). The accuracy and reproducibility of MRI enabled us to detect small changes in LV and septal mass as early as one month after PTSMA (29). In our study, the reduction in total LV mass significantly exceeded the reduction in septal mass, and the reduction in remote myocardial mass proved statistically significant. This may be explained by the reduction in LVOT obstruction that may have caused early regression of (secondary) LV hypertrophy by decreasing LV pressure and wall stress. An alternative explanation could be that ethanol infusion induced infarcts in other parts of the LV wall, by distributing through the capillary network. However, we found no evidence of induced myocardial infarction outside the target area, despite the very sensitive nature of CE MRI to visualize discrete microinjury (30).

The septal infarction caused wall thinning and loss of regional wall thickening, which led to a small, but

significant, increase in LV end-systolic volume. Left ventricular end-diastolic volume was unchanged, and, as a result, ejection fraction slightly decreased. Although the changes were only small, they illustrate again the effects of ethanol-induced infarction. Further study is necessary to evaluate the long-term effects of PTSMA on LV mass and volumes.

Study limitations. The total number of patients in our study group was limited. Three patients (11% of the initial study group) could not undergo follow-up MRI because a procedure-related atrioventricular block necessitated pacemaker-implantation. Infarct size according to peak CK-MB in these three patients was similar to the study group (148 ± 29 vs. 198 ± 84 U/l, p = NS), suggesting that infarct location may be an important factor for the development of conduction abnormalities.

The foci of patchy hyperenhancement that were demonstrated in patients with HOCM may have interfered with determination of the infarction size. Generally, they would have caused an overestimation of the infarction size, by overlap of pre-existing foci and ethanol-induced infarction. However, it is unlikely that these areas have significantly influenced our results, because the size was small relative to the procedure-related infarction size.

Clinical implications. At present, the optimal size and location of the myocardial infarction with respect to clinical outcome is not known. Theoretically, the objective is to abolish the LVOT gradient by inducing an infarction in the basal septum with the smallest possible amount of myocardial damage, located at the site of maximal mitral-septal contact. A catheter position proximal in the target septal perforator artery may increase the success rate of PTSMA, but should be weighted against the possible risks of proximal balloon inflation, such as injury inflicted on the left anterior descending artery or the potential of slippage of the inflated balloon with retrograde ethanol leakage.

During PTSMA, temporary balloon inflation and selective coronary myocardial contrast echocardiography are used to probe the risk area (14,23). Although contrast echocardiography is helpful in selecting the target location of ablation and septal branch, it does not allow transmural evaluation of the myocardium and may, therefore, not be able to reliably predict the adverse occurrence of right-sided septal infarction. Additional studies are needed to explore the relation between the area at risk as estimated by contrast echocardiography and the ultimate size and location of the inflicted septal infarction.

In conclusion, we found that CE MRI was an excellent technique for the quantitative evaluation of septal myocardial infarction induced by PTSMA. The correlation between size and location of the infarction and early outcome provides important feedback and may help to optimize this promising therapeutic option in patients with HOCM.

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